necessary dose required varied in different individuals. The effect was maintained from 6 to 8 hours, and occasionally some effect was observed for the succeeding 24 to 48 hours. A definite effect on the electrocardiograms in certain cases after potassium administration was observed. Certain ill effects are occasionally noted, namely, gastric distress and diarrhea. The acetate and citrate caused less distress than the chloride or iodide. The paroxysmal nodal tachycardia observed in one case and the epistaxis and petechiae in another may have been due to potassium administration.

Conclusion. Potassium salt administration by mouth is effective in checking auricular and ventricular ectopic beats and tachycardia in the majority of cases of organic heart disease. The failure to affect arrhythmias in patients without other evidence of cardiac pathology may be used to differentiate this group from the former. Potassium administration does not prevent the occurrence of auricular fibrillation.

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The Relation Between Acidosis and Glucose Tolerance.

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In earlier work[1] it was shown that phlorhizin glycosuria the glucose tolerance was directly related to the nutritional condition of the animal. Low tolerance was noted in those animals which had been fasted and phlorhizinized for 5 to 6 days while the opposite was found in those dogs which had received large doses of carbohydrate 12 to 15 hours previous to the test, although these animals were still phlorhizinized. These variations were similar to those found in the same dogs in the non-phlorhizinized state after fasting or after feeding large amounts of carbohydrate before carrying out the glucose tolerance test.

In each case the increased tolerance is associated with an abolition or a considerable decrease in the ketosis. The present investigation was designed to determine whether the acidosis *per se* was the cause of the decreased ability to store carbohydrate. Since it is impossible to produce a ketosis and at the same time have a large carbohydrate storage, an acidosis was produced by the injection or

[1] Deuel, H. J., Jr., *J. Biol. Chem.*, 1930, in press.

oral administration of 10 to 15 gm. of NH_4Cl. Simultaneously with the introduction of the NH_4Cl, large quantities of carbohydrate food were given. Glucose tolerance tests were made on the 2 following days using 16 gm. of glucose as was the practice in the earlier experiments with which these results are compared. The CO_2 combining power of the blood plasma was reduced from the normal value of 55 to 50 volumes percent to as low as 15 volumes percent in one case. Six tests were made on 3 dogs and these all showed a very small rise in the blood sugar with a rapid return to the pre-glucose level. These results are identical with those obtained on the same animals in the well fed condition whether normal or phlorhizinized. It is concluded that acidosis is not a primary cause of decreased glucose tolerance.

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Does Copper Poisoning Produce Pigmentation and Cirrhosis of the Liver?

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Mallory, Parker and Nye[1] demonstrated pigmentation and cirrhosis of the liver in rabbits following administration of copper salts in their feed. Hall and Butt[2] repeated and extended the work of Mallory. They obtained a similar pigmentation and cirrhosis of the liver in rabbits by feeding copper acetate. In addition they made chemical analyses of the livers and demonstrated the presence of large quantities of copper. Flinn and VonGlahn[3] repeated many of Mallory's experiments and added others of their own. They concluded that copper salts and powdered metallic copper administered in the feed do not cause deposits of pigment in the livers of rabbits, rats and guinea pigs; nor do these substances produce cirrhosis. They maintained that animals fed on an exclusive diet of carrots developed a pigmentation comparable to that produced by Mallory and others. Polson,[4] in Great Britain, obtained results substantiating those of Flinn and VonGlahn.

[1] Mallory, F. B., Parker, Frederic, Jr., and Nye, R. N., *J. Med. Res.*, 1921, **42**, 461.

[2] Hall, E. M., and Butt, E. M., *Arch. Path.*, 1928, **6**, 1.

[3] Flinn, F. B., and VonGlahn, W. C., *J. Exp. Med.*, 1929, **49**, 5.

[4] Polson, C. J., *Brit. J. Exp. Path.*, 1929, **10**, 241.